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Calcium Cerebral Emboli, A Diagnostic Challenge: Two New Reports

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Abstract

Calcium cerebral emboli represent an unusual aetiology of ischemic strokes. However, they are not the only cause of intracranial intravascular calcium density images, as far as complicated atheromatous plaques have a very similar appearance and typical localization (i.e. the proximal middle cerebral artery). Nonetheless, an accurate identification of both types of intravascular calcium images in the set of an Acute Stroke Code is very relevant, given that the most appropriate therapy is different in each case as a consequence of their distinct underlying physiopathology. We present two cases of ischemic stroke secondary to calcium cerebral emboli, one of which undergone endovascular treatment, and we discuss the keys for the recognition of this entity as well as its management.

Keywords: *Calcium cerebral emboli, Complicated intracranial atherosclerosis, Acute ischemic stroke*

Introduction

Calcium cerebral emboli are quite rare, having an estimated prevalence of 3% among stroke patients. They are considered unusual emboli in recent classifications, like are air, fat or tumour cells emboli [1]. Two cases are presented and the differential diagnosis of intravascular calcium images within cerebral arteries as well as its therapeutic significance is discussed.

Cases Reports

The first case was a 78-years-old, left-handed woman with antecedents of hypertension, valvular atrial fibrillation anticoagulated with acenocumarol with poor INR control and mitral mechanical prosthesis who presented with sudden language alteration of unknown time of evolution. The neurological examination revealed a moderate global aphasia and a mild left faciobrachial paresis (The National Institutes of Health Stroke Scale, NIHSS 7). An established right frontotemporal hypodensity was observed in the computed tomography (CT) scan and the CT angiography (CTA) showed a stop in the right middle cerebral artery (MCA) at M1 level, in the same point where a calcium density dot could be visualized in the simple CT (Fig.1A), being highly suggestive of a calcium cerebral embolism. The origin of this embolism is unknown, given that the patient had been operated on the mitral valve position with mechanical prosthesis replacement but also presented abundant calcified plaques in the aortic arch in the CTA. Neither fibrinolysis nor endovascular treatment were performed, and an established right frontal infarct covering the insular cortex was seen in a control CT a few days later (Fig.1B). Anticoagulation was reintroduced a week later and certain neurological improvement was assessed during the hospitalization. Nevertheless, the crossed aphasia persisted, thus precluding autonomous management at home and she was finally discharged to a rehabilitation centre.

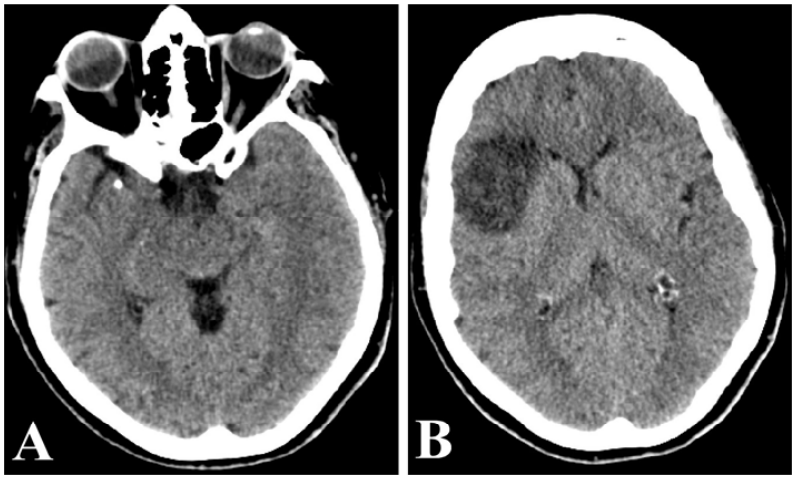

Fig 1. *Case 1. A. CT imaging showing a high density, round-shaped dot in the right M1 segment of the MCA compatible with a calcium embolus. B. CT imaging where an established infarct in the territory of the superficial branches of the right MCA can be appreciated. This lesion affected to the language area of this left-handed patient and produced a moderate crossed aphasia.*

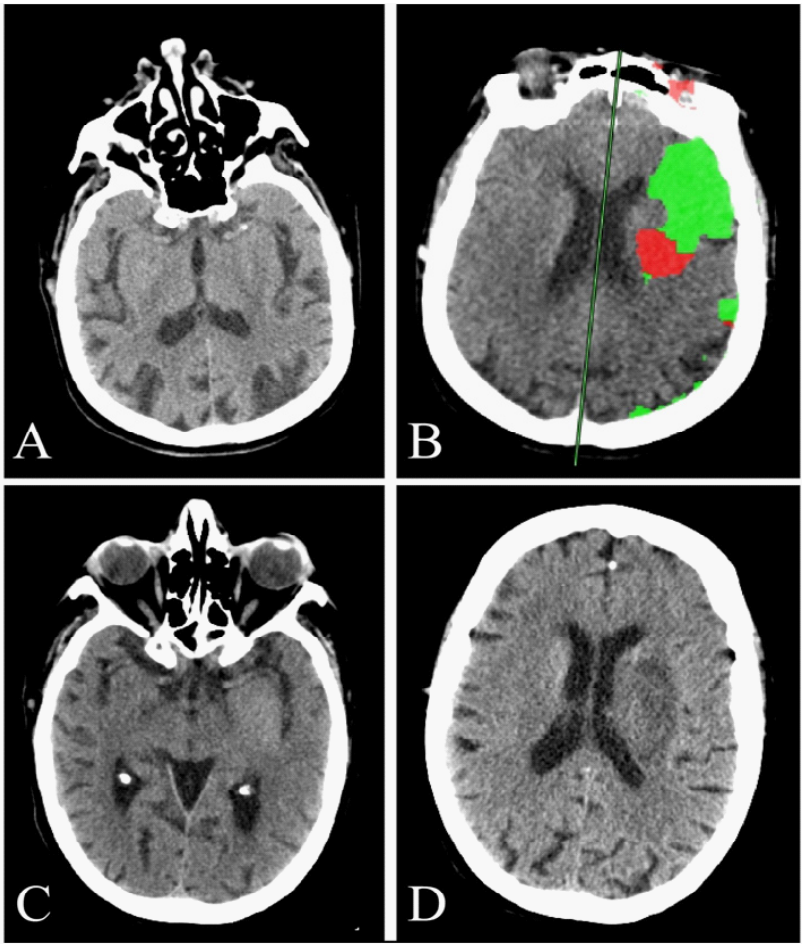

Fig 2. *Case 2. A. CT imaging showing a high density, round-shaped dot in the left M1 segment of the MCA compatible with a calcium embolus. B. CT perfusion imaging where a favourable mismatch between the core infarct (red) and the penumbra area (green) can be appreciated. C. CT imaging performed after the endovascular treatment demonstrating the vanishing of the calcium embolus. D. CT imaging displaying an established infarct in the territory of the deep branches of the left MCA.*

The second case was an 81-years-old, right-handed woman with antecedents of atrial flutter antiaggregated with aspirin 300 mg who presented with wake-up mixed aphasia, right hemiplegia and hemihipoestesia (NIHSS 15). The CTA showed a stop in the left M1 segment distal to a calcium density opacity (Fig. 2A) which was thought to correspond to an intracranial atheromatous plaque. Perfusion sequences were performed obtaining a favourable mismatch (Fig. 2B), therefore the patient undergone endovascular treatment and an embolus was removed through mechanical aspiration using an ACE™ 64 reperfusion catheter. Distal MCA twigs occlusions were seen in the final angiographic series (TICI 2b). The control CT demonstrated the disappearance of the calcium density dot (Fig. 2C), thus strongly suggesting the embolic nature of the occlusion. Calcified plaques in the aortic arch and carotid bifurcations were observed in the CTA, constituting a potential source of emboli. A control CT showed an infarct in the left basal ganglia (Fig. 2D). Anticoagulation with low weight heparin was begun, with posterior introduction of acenocumarol. Little early clinical improvement was assessed and the patient was finally admitted to a rehabilitation centre.

Discussion

We present two embolic strokes of presumable calcium nature, not totally demonstrable in the first case because of the lack of endovascular procedure, but confirmed in the second case given that the high density intravascular image vanished after mechanical thrombectomy. The most probable source of the calcium emboli in both cases was either the aortic arch or the carotid bifurcations, although a valvular origin could not be discarded in the first one, which is the most frequent provenience of this kind of emboli [2].

The main difficulty when assessing intracranial intravascular calcium density images lies in distinguishing complicated plaques from unusual emboli, and it poses a diagnostic and therapeutic challenge as far as in situ thrombosis due to a complicated plaque would beneficiate from either stenting or double antiplatelet therapy [3], while the best approach to arterial embolisms is mechanical thrombectomy, being highly controversial the use of intravenous fibrinolysis in the case of calcified emboli [4,5]. Some suggestive characteristics of emboli in comparison with in situ thrombi are high attenuation values (160 vs less than 70 Hounsfield Units respectively) and round shape in contrast to a linear profile [2]; and both features were met in the cases described here. Nonetheless, these keys do not provide enough certainty to allow making a confident decision about the best therapeutic strategy, even less during an Acute Code Stroke where time is scant and precious.

Thus, a careful examination of CT images seeking after intracerebral intravascular calcium density images in the set of acute stroke as well as the development of more confident keys for differentiating both types of intraarterial calcium, remain as a pending issue in the evaluation of Code Stroke patients, as far as it could condition the ulterior therapeutic approach.

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